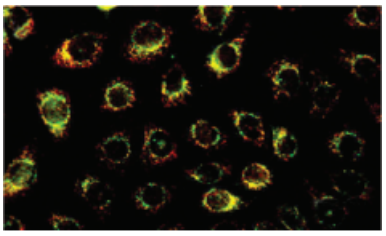
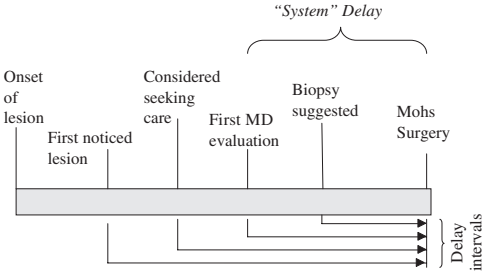
Keratinocyte Antimicrobials



Patients with atopic dermatitis are at greater risk for skin infections because they are unable to release more antimicrobial peptides during inflammation. Braff and coworkers found that lamellar bodies, the secretory, lipid-rich granules in the superficial epidermis, act as storage sites for keratinocyte cathelicidin. Showing that keratinocytes have the enzymatic machinery necessary to synthesize, store and activate this antimicrobial peptide confirms that the skin functions as an independent direct immune barrier. This furthers our understanding of the regulation of keratinocyte-derived antimicrobial peptides and may have an important role in the treatment of skin disease. J Invest Dermatol 124:394–400, 2005.

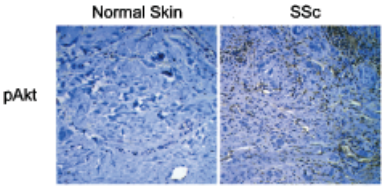
A Stitch in Time



Larger keratinocyte carcinoma (KC) lesions are associated with higher morbidity than less advanced disease. Eide and coworkers explored the association of potentially modifiable characteristics, including treatment delay, with KC defect size after Mohs micrographic surgery (MMS). More than one year's delay between initial physician examination and MMS was associated with misdiagnosis, failure to initially biopsy, and multiple surgical removals, and was ultimately associated with a doubling of defect size. Skin cancer information campaigns and screening programs presume that earlier diagnosis can minimize disease impact, but this study finds that attention to KC care delivery may be a more important factor in reducing morbidity. J Invest Dermatol 124:308–314, 2005.

Akt Activates SSc Fibroblasts

Transforming growth factor (TGF)-β plays a prominent role in the pathogenesis of systemic sclerosis (SSc), a chronic autoimmune, vascular and fibrotic disease. Fibroblasts from patients with SSc are relatively resistant to apoptosis and TGF-ß activates the kinase Akt, which has powerful anti-apoptotic effects. Therefore, Jun and colleagues examined whether Akt was activated in skin fibroblasts of SSc patients. They showed that, indeed, TGF-β signaling through Akt contributes to disease pathogenesis. Further study will need to distinguish whether additional factors, such as other cytokines, growth factors, or ligands such as CD40L, could also potentially activate Akt. J Invest Dermatol 124:298–303, 2005.



Vitamins for Sunny Days

DNA damage caused directly by UV radiation or indirectly by reactive oxygen species (singlet oxygen and free radicals) may lead to mutations and consequently, to skin cancer. Because antioxidants have been suggested as protective against UV-induced epidermal damage, Placzek and coworkers tested 18 volunteers with 3 months oral intake of vitamins C (2 g/day) and E (1000 IU/day). Results showed a protective effect against UVB-induced erythema. Significantly reduced thymine (cyclobutane pyrimidine) dimers in the skin post-treatment suggest this protocol protected against DNA damage. It remains to be seen whether antioxidants can also protect against skin cancer. J Invest Dermatol 124:304–307, 2005.

ß-Carotene: Versatile Molecule

UVA exposure is thought to cause skin aging mainly by singlet oxygen ($^1O_2$)-dependent pathways. ß-carotene (ßC) scavenges $^1O_2$ and other reactive oxygen species; reduces sunburn; and metabolizes to retinoic acid, a molecule involved in skin maintenance. Wertz and coworkers analyzed how ßC influences the response of irradiated HaCaT keratinocytes, which are exceptionally sensitive to UVA-induced apoptosis. Multiple genetic mechanisms were identified and included but were not restricted to $^1O_2$-quenching. These findings help explain conflicting reports on the efficacy of ßC as an antioxidant and have implications for treating photoaging as well as diseases such as skin cancer and psoriasis. J Invest Dermatol 124:428–434, 2005.